HALL (J. N.)

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A CASE OF DOUBLE PULMONIC MURMUR, WITH DIASTOLIC THRILL.

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WELL-MARKED disease of the pulmonary valves in adult life is sufficiently infrequent to make the following case worthy of record:

Mrs. R. M——, twenty-four years of age, with no family history of importance in this connection, presented herself at the medical clinic of the University of Colorado six months ago. She had one healthy child, twelve months of age; no other labors. Although subject to severe attacks of bronchitis for some years, especially in winter, her babyhood and childhood, with the exception of whooping-cough, were apparently free from serious disease. There was no history of rheumatism.

She complained of cough, expectoration, and dyspnœa, and presented high temperature and rapid pulse, with moderate dulness and moist râles in both bases, especially behind. I examined the heart, with negative result. The patient was sent to her home and treated by Miss C. L. Moore, a senior student, she reporting to me upon the progress of the case, which I did not see again. Although the dyspnœa was very marked, recovery ensued after a febrile course of about ten days. The case, although not typical, was regarded as one of pneumonia. Miss Moore examined the entire chest frequently during the illness, without noting anything unusual in the heart's action.

One month ago the patient applied for treatment for

a severe bronchitis, sonorous and sibilant râles being heard through the clothing with the unaided ear by the student detailed to the case. In verifying the student's report, I heard, in addition to the râles mentioned, a very loud and unusual murmur over the heart. The chest having been stripped to complete the examination, the following conditions were noted:

The patient was well developed, of natural color, with the exception of slight anæmia, and presented no œdema, lividity, or clubbing of the fingers. Weight, one hundred and thirty-five pounds. Temperature, normal; pulse, 100; respiration, 34 in a state of rest, but increasing in frequency upon slight exertion. Abundant rough, sonorous, and sibilant râles throughout the chest, without perceptible dulness. Cardiac dulness increased moderately upward and to the right, and nearly to the nipple line to the left. Apex beat just within this line in the fifth space. Valvular sounds of heart normal, excepting over the pulmonic region.

Here there was heard a very loud, whizzing, humming murmur, continuous, but accentuated at the beginning of systole, and changing in timbre at the time of occurrence of the normal second sound, which sound, however, could not be made out over the pulmonic area. The murmur, after the change of timbre noted, continued, louder than before, until the next systole. There was not the perfectly distinct intermission between the two parts of this murmur heard in double murmurs at the aortic and the mitral valves. The systolic portion of this murmur was transmitted much less distinctly, especially downward, over the precordial area, than the diastolic, but was heard with great distinctness between the left scapula and the spinal column, where the diastolic murmur was inaudible. The diastolic portion of the murmur was transmitted most strongly downward, especially toward the apex, but was heard quite distinctly over the entire precor-

dia. Over the second interspace, covering an area of about the size of a silver dollar, was a purring thrill, very distinctly diastolic and occupying the entire diastole.

The bronchitis disappeared after about two weeks, and the patient was exhibited at the meeting of the Denver and Arapahoe Medical Society, March 23, 1897. She was examined by many members of the society, and there was no dissent as to the presence of the signs described, although some discussion occurred as to their interpretation.

My own theory as to the origin of the disease and the interpretation of the signs is as follows, viz.: that she suffered for years previously, possibly from birth, from some defect in the pulmonary valve not giving rise to distinct physical signs. The frequent and severe attacks of bronchitis furnish ground for such a suspicion. The attack of pneumonia, a disease which is prone, as we know, to light up an endocarditis, may well have caused a sudden increase in such a valvular trouble if of endocarditic nature, or may have originated an endocarditis *de novo* upon a congenitally deformed valve. At least, the rapid development of the physical signs since the pneumonia would certainly lead one to connect this disease with the valvular trouble.

The systolic portion of the murmur, rough and transmitted into the back, leads me to infer a narrowing of the pulmonic orifice, while the diastolic portion, with distinct thrill, leaves no room for doubt, I believe, as to the existence of pulmonic insufficiency.

The only case comparable to this one which has come under my observation I saw but once, the woman suffering at the time from an attack of colic. She was thirty years of age, and had experienced, ten years previously, a very severe attack of measles. Three months before she had nearly died from an attack of

puerperal fever following an instrumental labor; and she also gave a history of a mild attack of acute rheumatism. So far as the organs within the chest were concerned, she complained only of palpitation and dyspnœa upon exertion.

The area of cardiac dulness was slightly enlarged, and she presented the following murmurs, without thrill, viz.:

(*a*) An apical systolic murmur, transmitted into the axilla, having the usual characteristics of the murmur of mitral regurgitation.

(*b*) A diastolic murmur, heard most distinctly over the aortic area, transmitted toward the lower end of the sternum and slightly to the left, prolonged, and rather rough in character.

(*c*) A diastolic murmur, soft and blowing, most distinct at the second left interspace, of different timbre from the one to the right of the sternum, transmitted directly downward to the fourth rib.

Between the locations of the two last-described murmurs was a space where neither could be heard distinctly. I do not feel, however, in the absence of thrill, in the presence of well-defined aortic regurgitation, and especially in view of the known rarity of the affection, that one could speak positively of pulmonic regurgitation in this case, although I believe it existed. I considered it possibly of septic origin.

In the first case, Dr. Charles Denison suggested the possibility of pressure upon the pulmonary artery by tuberculous deposit in the lung, or by tuberculous glands. In a careful examination since the recovery from the bronchitis, I was unable to find any evidence, rational or physical, of such a condition.